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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/756,101	01/13/2004	Steven M. Dubinett	G&C 30435.152-US-I1	3604

22462 7590 02/27/2007

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EXAMINER	
UNGAR, SUSAN NMN	
ART UNIT	**PAPER NUMBER**
1642	

SHORTENED STATUTORY PERIOD OF RESPONSE	MAIL DATE	DELIVERY MODE
3 MONTHS	02/27/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

If NO period for reply is specified above, the maximum statutory period will apply and will expire 6 MONTHS from the mailing date of this communication.

Office Action Summary	Application No.	Applicant(s)	
	10/756,101	DUBINETT ET AL.	
	Examiner	Art Unit	
	Susan Ungar /	1642	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☐ Responsive to communication(s) filed on *27 November 2006*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☐ Claim(s) *1-32* is/are pending in the application.

4a) Of the above claim(s) *1-31* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☐ Claim(s) *32* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *5/12/04*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

1. The Election filed November 27, 2006 in response to the Office Action of September 25, 2006 is acknowledged and has been entered. Claims 1-32 are pending in the application and Claims 1-31 have been withdrawn from further consideration by the examiner under 37 CFR 1.142(b) as being drawn to non-elected inventions. Claim 32 is currently under prosecution.

2. Applicant's election with traverse of Group 16 claim 32 is acknowledged. The traversal is on the ground(s) that examination of all groups would not impose a serious burden on the examiner. This is not found persuasive because the literature search, particularly relevant in this art, is not coextensive and different searches and issues are involved in the examination of each group. For these reasons the restriction requirement is deemed to be proper and is therefore made FINAL.

Claim Rejections - 35 USC § 112

3. Claim 32 rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claim 32 is confusing because the claim is drawn to a method of obtaining "a dendritic cell from a mammal" and transducing said cell and placing the single cell generated at the site of tumor to attract a T lymphocyte or a mature host dendritic cell to the tumor. The claim is confusing because the Office interprets the modifier "a" to mean one and only one. However, it appears from the reading of the specification that multiple dendritic cells are transduced and that it is expected that the multiple modified dendritic cells will attract multiple dendritic cells and T-cells. Thus, it is not possible to clearly understand the metes and bounds of the patent protection claimed. It is noted that amendment of the claim,

for example, to delete the term "a" and to indicate that the it is T lymphocytes and mature host dendritic cells that are claimed to be attracted and to delete the term "a" and to indicate that dendritic cells are obtained would obviate the instant grounds of rejection.

Claim Rejections - 35 USC § 102

4. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.

5. Claim 32 is rejected under 35 U.S.C. § 102(a) as being anticipated by Kirk et al (2001, Cancer Research, 61:2062-2067).

The claim is drawn to a method of attracting a T lymphocyte or a mature host dendritic cell to a site of a syngeneic tumor in a mammal comprising obtaining a dendritic cell from the mammal, introducing an exogenous polynucleotide encoding SLC as shown in SEQ ID NO:1 into the dendritic cell so that the cell expresses SLC, placing the dendritic cell at the site of the syngeneic tumor in the mammal, wherein the SLC expressed by the dendritic cell attracts T lymphocyte or mature host dendritic cell to the site of the syngeneic tumor in the mammal.

It is noted that B6 mice are an inbred, that is genetically identical, species of mice, see product sheet from Taconic Farms, attached hereto as Appendix 1.

It is assumed, given the indefinite claim language, for examination purposes that the claim is drawn to modifying multiple dendritic cells and attracting multiple T lymphocytes or mature host dendritic cells.

Kirk et al specifically teach that SLC is known to recruit DC and T cells to tumors and specifically exemplifies the recruitment in vivo (see p. 2064-2065) and teach a method of isolating dendritic cells from B6 mice and modifying the dendritic cells (DC) to comprise polynucleotide construct encoding SLC (see p. 2063, col 2). The reference further teaches that the intratumor injection of SLC-expressing DC into established B16-BL6 melanoma tumors (derived from B6 mice), in the B6 mice, successfully reduces tumor size. The reference further exemplifies the migration of DCs and T-cells into tumor in modified DC treated B6 mice tumors but not control treated animals (p. 2065). The reference teaches that administration of the modified DCs intratumorally in B6 mice resulted in tumor growth inhibition that was significantly better than DC control or SLC alone. Although the reference does not specifically teach that the SLC is an SLC as shown in SEQ ID NO:1, the claimed SLC and the claimed method appear to be the same as the prior art therapeutic agents, absent a showing of unobvious differences. The office does not have the facilities and resources to provide the factual evidence needed in order to establish that the product of the prior art does not possess the same material, structural and functional characteristics of the claimed product. In the absence of evidence to the contrary, the burden is on the applicant to prove that the claimed product and method are different from those taught by the prior art and to establish patentable differences. See In re Best 562F.2d 1252, 195 USPQ 430 (CCPA 1977) and Ex parte Gray 10 USPQ 2d 1922 (PTO Bd. Pat. App. & Int. 1989). It is further noted that although Kirk et al do not

specifically state that the DCs were isolated from the treated mammal, given that all B6 mice are genetically identical, given that it appears that the limitation of "obtaining a dendritic cell from the mammal" appears to be a product by process limitation for obtaining syngeneic (that is genetically identical) dendritic cells, the instant reference anticipates the claimed invention because all B6 mice are genetically identical and the production of a product by a particular process does not impart novelty or unobviousnes to a product when the same product is taught by the prior art. This is particularly true when the properties of the product are not changed by the process in an unexpected manner. See In re Thorpe, 227 USPQ 964 (CAFC 1985); In re Marosi, 218 USPQ 289, 292-293 (CAFC 1983); In re Brown, 173 USPQ 685 (CCPA 1972).

Claim Rejections - 35 USC § 103

6. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

The factual inquiries set forth in *Graham* v. *John Deere Co.*, 383 U.S. 1, 148 USPQ 459 (1966), that are applied for establishing a background for determining obviousness under 35 U.S.C. 103(a) are summarized as follows:

1. Determining the scope and contents of the prior art.
2. Ascertaining the differences between the prior art and the claims at issue.

3. Resolving the level of ordinary skill in the pertinent art.

4. Considering objective evidence present in the application indicating obviousness or nonobviousness.

7. Claim 32 is rejected under 35 U.S.C. 103(a) as being unpatentable over WO/038706 (IDS item) or WO96/06169 (IDS item) in view of Kirk and Mule (Human Gene Therapy, 2000, 11:797-806), Nishioka et al (Cancer Research, 1999, 59:4035-4041), Miller et al (Human Gene Therapy, 2000, 11:53-65) and further in view of Lode et al (Drugs of Today' 2000, 36:321-336).

The claim is drawn to a method of attracting a T lymphocyte or a mature host dendritic cell to a site of a syngeneic tumor in a mammal comprising obtaining a dendritic cell from the mammal, introducing an exogenous polynucleotide encoding SLC as shown in SEQ ID NO:1 into the dendritic cell so that the cell expresses SLC, placing the dendritic cell at the site of the syngeneic tumor in the mammal, wherein the SLC expressed by the dendritic cell attracts T lymphocyte or mature host dendritic cell to the site of the syngeneic tumor in the mammal.

WO 00/038706 specifically teaches a successful method of inhibiting the growth of tumors/cancer, B16-BL6 melanoma tumors (derived from B6 mice), in BDF-1 mice *in vivo* comprising administering a therapeutically effective amount of SLC (see abstract), wherein the SLC is the SLC of Nagira et al (1997, JBC, 272:19518-19524) and thus identical to SEQ ID NO:1. The specification teaches that the invention is based on the discovery that SLC inhibits the growth of tumors and is chemotactic for mature dendritic cells (p. 6, lines15-20) as well as other immune cells including T cells (p. 13, lines 28-30) and the invention is drawn to a method of treating cancer in a mammalian subject comprising administering

polynucleotide encoding SLC, in situ (p. 12, lines 26-30 and claim 11) to a subject, wherein the SLC is preferable human SLC (p. 6, lines 24-29 and claims 1 and 2), wherein said administration is by injection into said tumor (claim 9), (p. 12, lines 6-15).

WO96/06169 disclose a method of treating solid tumors comprising the administration of a polynucleotide encoding Ckbeta-9 polypeptide of sequence identifier 2,which comprises human SEQ ID NO:1 of the instant invention (page 3, lines 28-34, page 18, lines 11-21). The reference further states that the chemokine treats the tumors by stimulating the invasion and activation of host defense cells, e.g. cytotoxic T cells and macrophages (p. 18, lines 11-15). The reference specifically teaches that cells from a patient may be engineered with a polynucleotide (DNA or RNA) encoding a polypeptide ex vivo, with the engineered cells then being provided to a patient to be treated with the polypeptide. Such methods are well-known in the art (p. 29).

The references teach as set forth above, but does not teach a method of treating syngeneic tumors, introducing polynucleotide encoding SEQ ID NO:1 into a dendritic cell from the mammal, so that the cell expresses SLC and placing the modified dendritic cell at the site of the syngeneic tumor in the mammal.

Kirk and Mule (Human Gene Therapy, 2000, 11:797-806) specifically teach that the conventional nature of genetic modification of DCs to express immunomodulatory proteins such as cytokines and chemokines, wherein the reference suggests that these modified DCs may be used as adjuvants to treat any number of tumors so long as a source of tumor antigens is available and further suggest that said modified DCs are more potent than gene-modified tumor cells since the modified DCs are both APC and cytokine factories while the tumor cells

require host APC function. Further, the reference specifically points to murine models of melanoma and sarcoma wherein DCs genetically modified to express immunomodulatory proteins such as cytokines and chemokines injected directly into tumors induced a profound anti-tumor effect (p. 802, col 1).

Nishioka et al (Cancer Research, 1999, 59:4035-4041) specifically teach that isolated bone marrow-derived dendritic cells (DCs) retrovirally transduced with genes encoding the cytokine IL-12 stably expressed bioactive IL-12 at high levels and that intratumoral injection of the modified DCs resulted in regression of day 7 established weakly immunogenic tumors when compared to treatment with IL-12 transduced fibroblasts or control DCs (see abstract) in B6 inbred mice (p. 4036 col 2), wherein primary culture of syngeneic fibroblasts were obtained from the lungs of B6 mice and BM-DC cultures were obtained. Considering the known antitumor effects mediated by local expression of IL-12 and the capability of DCs to induce an effective systemic immune response, as well as the known ability of DCs to take up and process tumor antigens makes them a superior choice for local cancer treatment immunomodulation compared to non-professional APCs such as IL-12 transduced fibroblasts (p. 4035, col 2). The reference goes on to teach that DCs pulsed with synthetic tumor peptides, as well as pulsed with tumor eluted peptides and tumor lysates, induce an effective antitumor immune response and that patients are now being treated with this construct. However, this approach will not be applied to the majority of patients due to technical difficulties and cumbersomeness of the preparation of these materials from human solid tumors. An alternative approach is to deliver DCs directly to the site of the tumor. The authors have recently shown that DCs injected intratumorally are capable of capturing tumor antigens *in situ* at the tumor site and inducing a subsequent

systemic immune response against the tumor. Using this method, although an immune response was generated, this was not sufficient to eradicate preexisting vigorous tumors. The authors hypothesized that genetic modification of the DCs would enhance the systemic antitumor response to a level sufficient for effective treatment and demonstrate that intratumoral injection with IL-12 gene-modified DCs is capable of significantly suppressing the growth of established tumors and were more effective that IL-12 transduced syngeneic fibroblasts.

Miller et al teach that intratumor administration of IL-7 transduced dendritic cells into B6 mice presenting with lung cancer resulted in complete tumor regression. Comparison with other intratumoral therapies including recombinant IL-7, IL-7 vector alone, unmodified DCs, IL-7 transduced fibroblasts or DCs pulsed with tumor lysates revealed that DC AdIl-7 therapy to be superior in achieving antitumor responses and in augmenting immunogenicity (see abstract). Further, in those that mice had complete tumor eradication as a result of either CD-ADIL-7 or AdIL-7 therapy, when rechallenged with parental tumor cells 30 days or more after complete tumor eradication, all of the DC-AdIL-7 treated mice completely rejected a secondary rechallenge, whereas the AdIL-7 treated mice had sustained antitumor effects in only20-25% of the mice (see abstract). The authors note that the in situ administration of the construct lead to utilization of the tumor as an in vivo source of antigen for DCs. In contrast to in vitro immunization with purified peptide Ag, autologous tumor has the capacity to provide the activated DCs administered at the tumor site access to the entire repertoire of available antigens in situ. The authors report that intratumor injection of DC-AdIL-7 is effective in eradicating established tumors and generating systemic antitumor immune responses (p. 54, col 1). The reference discloses the isolation of DCs from

bone marrow and the transduction of the DCs with AdIL-7 (p. 54, col. 2).

The authors teach that the problems in utilizing tumor antigen-based immunization strategies include (1) the potential induction of tolerance, (2) the inability to utilize repeated dosing because of vector-associated neutralization and (3) the limitation of therapy to patients whose tumors express defined specific tumor antigens in the context of the correct HLA phenotype. They now describe a therapeutic paradigm that overcomes these deficits. This anti-tumor DC-based therapy exploits the professional APC as an effective vehicle for cytokine delivery and presentation of multiple tumor antigens *in situ*. The results of this study demonstrate the capacity of intratumoral DC-AdIL-7 therapy to eradicate established murine lung cancers in two different models. The authors state that the DC-based intratumoral delivery of an immunopotentiating cytokine results in potent systemic, specific antitumor immune responses and heightened immunogenicity (p. 61).

Lode et al (Drugs of Today' 2000, 36:321-336) teach that it was a common strategy in immunotherapy of cancer, at the time the invention was made, to assay immunomodulation of syngeneic malignancies. This report summarizes successful therapeutic efficacy and immune mechanisms involved in targeting the chemokine Il-2 to syngeneic tumors in syngeneic animal models (see abstract) wherein it is taught that targeted chemokine was effective for treating syngeneic cancers, wherein for example, the cancer cells were non-spontaneous, experimental as well as spontaneous cells which metastasized, for example in the Neuroblastoma model to sites typical for human neuroblastoma, including bone marrow, liver, lymph nodes and adrenal glands (p. 328, col 1), wherein in numerous examples, the inhibition of growth of the syngeneic tumor cells is measured by quantification of

tumor surface area (see entire reference) but that untargeted chemokine was not effective for treating syngeneic cancers (see for example p. 327, col 1).

It would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made to have substituted the SLC construct of either WO/038706 or WO96/06169 for the constructs of any of Kirk and Mule, Nishioka et al, Miller et al for the preparation of modified DCs which express immunomodulatory proteins such as cytokines and chemokines in order to treat the cancers of Kirk and Mule, Nishioka et al, Miller et al because Kirk and Mule, Nishioka et al, Miller et al because the cited references teach successful conventional methods of transducing dendritic cells with polynucleotides encoding immunomodulatory proteins and the advantages of their use in the treatment of cancer. In particular, (1) Kirk and Mule specifically teach that the modified DCs may be used as adjuvants to treat any number of tumors and specifically teach the advantages of using modified DCs as opposed to gene-modified tumor cells wherein the gene modified DCs are both APC and cytokine factories while the tumor cells require host APC function, In addition, (2) Nishioka et al specifically teach that the combination of known antitumor effects mediated by local expression of cytokine (for SLC that is the attraction of DCs and T-cells) and the capability of DCs to induce immune response, as well as the known ability of DCs to take up and process tumor antigens makes them a superior choice for local cancer immunomodulation treatment which is a treatment superior to even effective treatment with DCs pulsed with synthetic peptides, tumor eluted peptides or tumor lysates which have been shown to induce effective antitumor immune response. The reference further teaches that the cytokine modified DCs are superior because the DCs pulsed with synthetic peptides, tumor eluted peptides or

tumor lysates cannot be applied to the majority of patients due to technical difficulties while the immunomodulatory technique can be broadly and successfully applied to many different patients with many different types of cancer. Finally, the reference teaches the improved efficacy of transduced gene-modified DCs which comprise polynucleotides encoding immunomodulatory proteins compared to even transduced gene-modified fibroblasts comprising polynucleotides encoding immunomodulatory proteins. (3) Miller et al specifically teach the advantages of cytokine modified DCs in the treatment of cancer and demonstrate the improved efficacy of transduced gene-modified DCs which comprise polynucleotides encoding immunomodulatory proteins over the administration of vector comprising polynucleotides encoding immunomodulatory proteins.

One would have been motivated to substituted the SLC construct of either WO/038706 or WO96/06169 for the constructs of any of Kirk and Mule, Nishioka et al, Miller et al for the preparation of modified DCs which express immunomodulatory proteins such as cytokines and chemokines in order to treat the cancers of Kirk and Mule, Nishioka et al, Miller et al because although both WO/038706 or WO96/06169 teach the method of treating tumors with immunomodulator that attracts DC and T-cell to tumor and WO/038706 specifically exemplifies successful treatment, Kirk and Mule, Nishioka et al, Miller et al teach the advantages of using cytokine gene modified DCs over other polynucleotide constructs for the local administration and expression of cytokines. Given the importance of treating cancer, motivation is clear for using the method of the combined references to further improve efficacy of treatment.

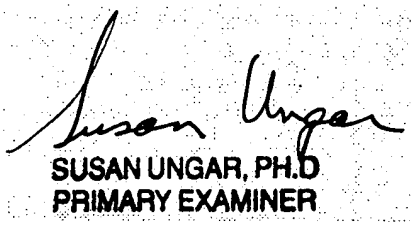
Finally, it would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made to have substituted the SLC DC construct of the combined references in any of the methods of the reviewed syngeneic experiments in Lode et al, and to use isolated syngeneic DCs to avoid immune response against the DCs, because Lode et al specifically teach that it was a common strategy at the time the invention was made to assay immunomodulation in syngeneic malignancies and discloses the use of syngeneic animal models to determine the efficacy of treatment. Further, it would have been *prima facie* obvious to one of ordinary skill in the art to substitute the SLC construct of the combined references into the method of any of the reviewed experiments in Lode et al because all of experiments are drawn to immunomodulation by cytokines and SLC is a cytokine known to be an immunomodulator, thus SLC is a functional equivalent of the cytokines of Lode et al. In view of the functional equivalence, one would have a reasonable expectation of success in substituting the SLC construct of the combined references for the constructs disclosed in Lode et al.

8. No claims allowed.

9. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Susan Ungar, PhD whose telephone number is (571) 272-0837. The examiner can normally be reached on Monday through Friday from 7:30am to 4pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Shanon Foley, can be reached at 571-272-0898.. The fax phone number for this Art Unit is (571) 273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).



February 8, 2007

Appendix I

Products & Services

- Contract Breeding Solutions
- **Mouse & Rat Models**
  - Transgenic Models
  - **Traditional/Spontaneous Mutant Models**
  - Custom Hybrid
- Induced Mouse & Rat Models
- Genetically Modified Models
- Contract Research Solutions
- Testing Services
- Surgical Modifications
- Animal Husbandry Products
- Shipping Products

MOUSE & RAT MODELS

Black 6

TRADITIONAL INBRED MICE

C57BL/6NTac Background

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+45 70 23 04 05 (E

Model Number	Zygosity	Nomenclature
B6-F		C57BL/6NTac
B6-M		C57BL/6NTac

Description | Growth Chart | Health Reports/Sites | Phenotyping | Price T

Model Description

C57BL/6 inbred mouse, from NIH 1991 at F151

Origin: C57BL/6 litters were received in 1991 at F151 from the NIH Animal Genetic Resource. Cesarean d 1991 at Taconic, a foundation colony is maintained in gnotobiotic isolators. Origin is as follows: to NIH in 1 Jax at F32; to Jax in 1948 from Hall.

Color: Black

Coat Color Loci: a

Genetics:

- *Strain Profile*: $Acy1^{f}$, $Alad^{b}$, $Car2^{a}$, $Cas1^{g}$, $Cd5^{b}$, $Cd72^{b}$, $Cd8a^{b}$, $Cd8b1^{b}$, $Ce2^{a}$, $Es1^{a}$, $Es3^{a}$, Esd^{a}, Ggc Gus-s^{b}, $H6pd1^{b}$, Hba^{a}, Hbb^{s}, Hc^{1}, $Idh1^{a}$, $Mod1^{b}$, $Mup1^{b}$, $Pep3^{a}$, $Pgm1^{a}$, Trf^{b}
- *Immunology*: $H2^{b}$, $Ptprc^{b}$, $Thy1^{b}$, $H2\text{-}T18^{b}$

Animal Diet: NIH #31M Rodent Diet